conservative. The third method described is liberal in ample food, somewhat radical but apparently very effective. The fourth method described is a safe and successful procedure of therapy. The third method, therefore, is, in my opinion, the best because it affords the possibility of reducing immediate mortality from gross hemorrhage by liberal feeding.

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CHRONIC NONHEALING LESIONS OF THE NOSE*

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REPORT OF CASE

CASE 1.—Miss C., a white woman of 40, came to me in July, 1935, because of discharge from the left side of her nose. During the past year she had been to several ear, nose, and throat men, and had not been relieved. Examination of the nose showed slight irritation in the left vestibule, which extended onto the inferior turbinate a short distance. The mucous membrane looked rather punched out. There was a small amount of thin discharge. X-rays of the sinuses were negative. I treated the nose locally for about a year and there was only a little improvement. The patient went East for a year and was treated by another ear, nose, and throat man; but her nose was never clear of irritation. In December, 1937, she returned to me. Examination then showed the original lesions on the inferior turbinate and vestibule completely healed; but on the septum, at the mucocutaneous junction, there was a reddened indurated area, nearly one-quarter inch in diameter, with a small ulcerated center. I cauterized this area, and treated it locally and with ultra violet light. As the lesion on her septum got worse instead of better, in March, 1938, I removed a liberal piece of the tissue, which was diagnosed tuberculosis. Instead of getting worse, this area healed up within a short time and has remained healed. The skin of the left side of the vestibule, however, again became irritated and has remained so since. Recently, when the human, bovine, and avian tuberculin tests were all done at the same time, this irritation in the vestibule flared up, but has since quieted down. Guinea pig check on the tissue was not done. The x-rays and examination of her chest are essentially negative. The x-rays of her bones show no sarcoid lesions. The Wassermann and other tests are negative. The tuberculin tests were all strongly positive.

TUBERCULOSIS OR SARCOID

Last summer, shortly after her biopsy had shown tuberculosis, I heard Dr. Frank Kistner of Portland, Oregon, give his paper at the American Medical Association, in which he reported a case of sarcoid of the nose. I then wondered if my case might come under that classification. I talked to Doctor Kistner about it, and sent him a microscopic section. He wrote me that it might be a sarcoid.

As I had never heard the term sarcoid before, I began to investigate this subject. I found that there is a very interesting controversy as to whether sarcoid is a form or phase of tuberculosis or not. There is extensive literature upon this subject, but to me there was one outstanding article: "Noncaseating Tuberculosis," written by Dr. Max Pinner and published in the *American Review of Tuberculosis* for June, 1938. It is a thirty-page article in which he analyzes the literature to date. He lists 217 references. He is a recognized authority and writer on tuberculosis and its pathology. To me his reasoning and conclusions are the clearest and most logical. He believes that tuberculosis and sarcoid are simply different manifestations of the same morbid process, and that the etiology in both is the tubercle bacillus.

Pinner feels that all the various forms of sarcoid are essentially the same disease which overlap each other clinically. Boeck, in 1899, was the first to use the term "sarcoid." Typically Boeck's sarcoid included involvement of the skin and mucous membranes, and lymph nodes. Gradually new regions were found involved and given the name of a new disease, such as Jüngling's disease, involving the bones; the uveoparotid fever, involving the uveal tract and parotid gland; and Mikulicz's syndrome, involving the lachrymal and salivary glands. There are many other combinations and forms that have been described and named, which I will not try even to tabulate; for, as Pinner says: "The terminology of Boeck's sarcoid and related lesions is confusingly and unnecessarily involved, particularly regarding the dermatological morphology, and the main difference is one of localization."

Probably the most important evidence that all sarcoids belong in one group is their uniform histology, whose unit is the epithelioid tubercle without caseation. Another factor is that most sarcoids have tuberculin anergy, which means "abnormal inactivity," and in the case of sarcoids it means negative tuberculin skin tests.

PATHOLOGY

The etiology of sarcoid is a controversial subject. Although there are a few men who believe that leprosy, rhinoscleroma, etc., are the cause of sarcoid, the greatest controversy is between those who believe it is due to the tubercle bacillus and those that do not. Pinner lists eight reasons why he thinks it is due to the tubercle bacillus:

1. Kyrle found the tubercle bacillus in the tissue during the first ten days of the lesion, but not afterwards.

2. The histological picture of sarcoid is characteristic of the noncaseating phase of tuberculosis.

3. Cases of sarcoid sometimes develop into classical tuberculosis.

4. Tuberculin anergy, he says, may be due to the skin itself, or to substances in the blood serum which neutralize the skin reactivity to tuberculin tests.

* Read before the Section on Eye, Ear, Nose and Throat of the California Medical Association at the sixty-eighth annual session, Del Monte, May 1–4, 1939.

5. Serological data (anticutins and procutins).

6. Sarcoid lesions disappear with the development of frank tuberculosis.

7. At the same time sarcoid lesions appear, other lesions may occur that are definitely or probably tuberculosis.

8. There is a close similarity between sarcoid reactions in humans and in certain animal species.

Pinner feels that all the sarcoids should be called noncaseating tuberculosis. The term "noncaseating" tuberculosis should not be understood as a separate type of tuberculosis, but rather as a *phase of tuberculosis;* and while usually chronic or stable, they may transform into a caseating form. Some lesions show minimal caseation; others more advanced. "There is no rigid dividing line between caseating and noncaseating phases; the typical representatives are clear-cut and easily recognizable, but transformation from the noncaseating to the caseating form and the border-line cases occur occasionally. The particular conditions that favor persistence of the noncaseating lesions are insufficiently understood. Failure to react to tuberculin is commonly associated with the noncaseating phases of tuberculosis. Whether this anergy is cause or effect, whether it is dependent on the infecting strain, on the constitutional or on immunological peculiarities of the host, or on the modalities of the apparently intermittent hematogenous dissemination, all these questions must wait for further studies to be solved."

INCIDENCE

Tuberculosis and sarcoid lesions of the nose are surprisingly rare in this region. I have asked many ear, nose, and throat specialists, as well as men specializing in diseases of the chest and tuberculosis, and I have written to several large sanitaria asking how many cases of tuberculosis of the nose they had had. Other than the three cases I am reporting today, I have found very few proven cases.

Sarcoid lesions of the nose are also rare. As sarcoid is a disease reported mostly by dermatologists, I have asked several of them how many cases they have seen. Their usual answer is that they have seen many cases; but when you limit it down to cases of sarcoid in the nose proven by biopsy, it becomes a rarity. There are at least two cases on the Pacific Coast reported in the literature. One is the case I previously mentioned of Dr. Frank Kistner. That report appears in the *Journal of the American Medical Association* for November 26, 1938.

The other case was reported by Dr. F. G. Novy and Dr. Nelson Keeler of Oakland in CALIFORNIA AND WESTERN MEDICINE for July, 1936. As they were kind enough to let me examine the patient recently, I wish to give a brief progress report on this case. She originally complained of nose-bleeds and nasal discharge. Several months later she had skin nodules. Biopsies were then taken from her nose and from skin nodules which were diagnosed sarcoid. Tuberculin tests were negative. At that time she had no x-ray evidence of bone lesions, but since that report she has developed five different bone lesions. One involves the terminal phalanx of a finger, where the bone is almost completely destroyed and the end of the finger is swollen to double its size. It is reddened and has drained a small amount, but is not very tender.

Following a septum operation she developed a perforation of the hard palate into her left nares at the site of one of these nodules. This has practically closed at present. There is no perforation of the septum. There is still marked crusting in both nares.

Besides the case report given at the beginning of this discussion, I wish to report the following two cases of tuberculosis that we have had at our office:

REPORT OF CASES

CASE 2.—H. G., a white man of 35, had had an external frontal operation done, at San Quentin, for an acute sinusitis. Following this, he had several flare-ups and fistulae that had been incised, necessitating reopening of the wound. In December, 1927, after a bad flare-up, he was again reoperated. Some tissue was removed from the ethmoid and turbinate region which showed typical tuberculosis. The external wound healed normally, but it was very slow. He went to Arizona later, as he had lung involvement. Two years later he was seen again, and the nose was in very good condition, externally and internally.

* * *

CASE 3.—Miss R., a white woman of 30, had a simple mastoid operation in 1929, and during a normal short convalescence she complained of nasal discharge. Examination showed a small granular mass, the size of a pea, on both sides of the nasal septum about one inch from the anterior tip. Microscopic sections of this tissue showed typical tubercles. No check of tissue was made on the guinea pig. Repeated sputum examinations were negative. X-rays of the chest showed considerable scarring, and calcification in the right lung and hilum as well as the cervical and axillary regions. These x-rays were repeated three years later, and again in April, 1939, and showed no essential change. X-rays of the bones showed no involvement. Tuberculin tests were not done in 1929; but two weeks ago the human and bovine tuberculins were strongly positive and the avian was mildly positive.

I treated the original lesions by removal of tissue for biopsy and cauterization with chemicals for about a year. The nose of the patient improved, but did not heal. She was not seen again until 1934. The nose was the same as before, but there was no extension of the process. Another biopsy again showed tuberculosis. I treated it as I had before, and also gave a long course of ultra-violet light treatment for eight months. It improved but did not heal. She moved from San Francisco and was not seen again until April, 1939. To my surprise, the nasal septum had completely healed, and there was no perforation of the septum, while the mucous membrane was perfectly smooth. However, the anterior tip of the left inferior turbinate looked somewhat granular to me. There was no ulceration, but, because of the previous lesions, I took a biopsy of this tissue. Again I was surprised, as it also showed tuberculosis.

TREATMENT

There is practically nothing in the literature on treatment. I tried every medication I knew of, including ultra-violet light treatments locally. None of them were effective. However, all three cases that I have treated healed at the site where the biopsy was taken. The two cases on the septum healed without perforation of the septum. Therefore, in the future, if I had a small localized lesion that I was suspicious of, or knew was tuberculosis, I would excise it.

IN CONCLUSION

We all know that there are various infections, such as syphilis, leprosy, glanders, rhinoscleroma, infected foreign bodies, and some forms of malignancy or new growth, that may produce lesions that look like tuberculosis. Also, the microscopic picture of these lesions may look like tuberculosis. These conditions must, of course, be eliminated in any differential diagnosis of a chronic nonhealing lesion of the nose. These other causes have been fully discussed at meetings and in the literature in recent years.

However, as the relationship of sarcoid to tuberculosis has not been discussed at our meetings, I felt that it would be worth while to limit my discussion to this subject. It is my belief that sarcoid is not a separate disease, but should be considered as a phase of tuberculosis.

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IMMUNITY: CLINICAL AND EXPERIMENTAL OBSERVATIONS*

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IMMUNITY is the topic of conversation by lay people almost as commonly as is the weather. At any bridge or tea will be heard such remarks as, "Johnny is just like his father; he catches everything that comes along. He has had measles three times. Susan never gets anything," etc. Many such popular beliefs, though not all, are supported by clinical observation; but when an attempt is made to measure immunity it remains one of the most intangible problems of science.

A cross section of a sample of the population at any age level should show a considerable variation in different individuals' ability to combat disease. Also a longitudinal study of any one person from birth to death should show a change in his resistance to infection or to any given infection at different times.

Even at birth may be revealed considerable variations in individuals' immune mechanism. At a delivery everyone is aware of the tension with which is awaited the first cry, indicating the establishment of respiration and circulation in the detached infant. The newborn has made a happy landing—so we think. There are, however, other functions than breathing and circulation which must become established, and one of these is the ability to live in an unsterile environment. Occasionally an infant in the nursery, even with normal heart and lungs, develops a breast abscess or pyelitis or even septicemia or meningitis. Frequently the causative organism is the ordinary staphylococcus or B. coli. A good doctor does, and rightly should, look for the source of infection in the environment—faulty technique, contamination of food, infection in an attendant, etc.—but in most instances he looks in vain, for the fault probably is within the baby himself. Some infants undoubtedly are inherently incapable of surviving bacterial invasion. Their bodies behave more like a culture medium than a living organism mobilizing to combat an invader. These babies simply turn sour and die.

If the newborn period is survived uneventfully, differences may manifest themselves later. A few years back a child died in our ward of measles encephalitis. The history of the boy brought out the interesting fact that he had had measles a few years previously. Second attacks of this disease are very unusual, and physicians of long experience will report only its rare occurrence. In this instance there was no reason to doubt the diagnosis because the same doctor had seen the child with both attacks, and in his records of the first was noted the presence of Koplik spots. Furthermore, the child had been vaccinated twice within a period of a few years, with a take each time. This, too, is unusual. He probably was a child with a defective immunizing mechanism. It is surprising he survived to childhood. At any age-level such differences manifest themselves. They should be less apparent later in life because of the toll along the way. Although modern care and precautions will safeguard many to later periods, yet the chances are great that some infection will catch up with them before they have traveled very far into time.

The longitudinal study of the individual is equally fascinating. Whether he has a good or poor resistance, his own reactions to a given infection may change. Thus, during the first few months of life he is immune to measles; this period is followed by increased susceptibility during the next year or two, and then during childhood the disease, if contracted, is not unduly severe. One attack confers immunity. In contrast to this, erysipelas, untreated, has a mortality of nearly 100 per cent in the newborn period. After that children withstand erysipelas well, and only in adult life, particularly old age, again is it a severe disease. It confers no lasting immunity.

To some infections the normal response is permanent immunity. This is true of chicken-pox, smallpox, measles, mumps, scarlet fever, and a number of other diseases. However, permanent immunity to the first infection of even these diseases is only relative. As pointed out earlier, authentic cases of second attacks of measles and chicken-pox are rare, but of whooping cough and scarlet fever are not uncommon. Within the last few years reinfections in poliomyelitis have been reported and ancient manuscripts record second attacks of smallpox. In other types of infection, such as tonsillitis, immunity is established gradually after repeated infections; attacks are frequent in childhood and gradually decrease with age. Other infections may make the individual more instead of less susceptible to repeated exposures. This appears to be the case with erysipelas and influenza. Still other organisms, notably the tubercle bacillus, produce entirely different manifestations in primary and secondary invasions. This latter characteristic has not been studied carefully in other diseases. It is possible that it manifests itself in rheumatic fever, nephritis, and others.

Immunity is not only intangible, it is at times capricious. Occasionally an individual is found

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